(5) Impairment of Gas Exchange and Dyspnea

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The concept of "dyspnea" is understood to include the perception and the reaction. The cause of the information is hardly known, but actually in many cases dyspnea is the most important cause of inactivity. Besides, even if it does not appear at rest, it is seen that dyspnea increases on exercise, by infections complicated, and at increased metabolism.

In this paper the relationship between various factors of dyspnea is at first considered from the standpoint of the impairment of gas exchange, then from the point of respiratory function, i.e., oxygen uptake and carbon dioxide output is discussed the hypothesis of length-tension inappropriateness, which is induced only from the pressure-volume relations of the chest.

Results:
1) Oxygen uptake and carbon dioxide output during exercise were recorded by the efficiency tester in cardiopulmonary patients with dyspnea.

The result showed that oxygen uptake as well as carbon dioxide output decreased as the degree of dyspnea increased.

2) Arterial blood gases and the work of breathing were studied on exercise in obstructive lung diseases.

When dyspnea was been conscious, arterial oxygen tension reduced to 57 mm.Hg and carbon dioxide tension rose to 55 mm.Hg. After exercise when dyspnea disappeared, two of them were returning to the initial.

When dyspnea appeared, the work of breathing had increased remarkably, then it disappeared, the latter seemed to return to the initial, and when hypoxemia was dismissed by breathing the pure oxygen, dysnea disappeared though the work left almost unchanged.

3) The alveolar function diagram was constructed in which the alveolar-arterial pressure differences of O₂ and CO₂, and the degree of dyspnea were plotted, in order to see the relationship between the impairment of gas exchange and the degree of dyspnea at rest.

Both of them showed proportional increase, and it was realized that the latent impairment of gas exchange was present at rest in patients with 2nd or 3rd degree of dyspnea, who were conscious of dyspnea only during exercise.

4) In addition, the relation was detected from the point of the efficiency of respiration. That is, all alveoli were divided into three or four groups of alveoli, then a
diagram was constructed in which ventilation and circulation per 1 cc. oxygen uptake and carbon dioxide output of every group of alveoli were plotted on the ordinate, and the ventilation-perfusion ratio on the abscissa.

Now, this is explained in the case of obstructive lung disease.

Compensatory hyperventilated alveoli may be within 10 per cent of all alveoli, but they are ventilated by the half of all and the ventilation-perfusion ratio of them is higher than 5.0. Ventilation per 1 cc. oxygen uptake of these alveoli increases as much as over five times of 14 cc. in normal alveoli. Consequently, ventilation to these alveoli, about half of all, is abandoned concerning oxygen uptake. While, about 70 per cent of all alveoli receive only 15 per cent of ventilation and their ventilation-perfusion ratio becomes 0.2 or 0.3 to befit poorly ventilated alveoli. Therefore, the unsaturated blood perfusing these alveoli may cause hypoxemia which stimulates ventilation very much.

Concluding the above-mentioned results, summarized blood gas disturbances based upon the impairment of gas exchange increase ventilatory drives of the respiratory center, and act on the lung-thorax system to increase the total ventilation, in order to supply with oxygen demanded. And in general, the work of breathing increases, and even if ventilation might increase, abnormally wide spread distributions of the ventilation-perfusion ratio would not be improved. Therefore blood gases could not be normal and would continuously cause ventilatory stimuli. In brief, it seems to this case that increased ventilatory stimuli do not effect on the respiratory function.

A few fundamental studies were performed to quantify the inappropriateness between them.

1) In normal subjects, ventilatory stimuli caused by blood gas disturbances may be reflected in expired minute ventilation. Ventilation and blood gas values of them were observed during exercise with the additional air way resistance and compared with the intact subject. Dyspnea appeared 2 min. 24 sec. on exercise when the air way resistance was added, on that time arterial oxygen tension was 56 mm.Hg and carbon dioxide tension 58 mm.Hg, while they changed little in the intact. Though minute ventilation of the former was only 10 L./min./m², which was less than 16 L./min./m² in the intact, the work of breathing increased much more than the intact.

2) To quantify the ventilatory drives caused by blood gas changes on that instance, changes in ventilation were observed under the low oxygen tension as the arterial carbon dioxide tension kept about 55 mm.Hg.

Ventilation increased intensely at the arterial oxygen tension below 70 mm.Hg, and at 56 mm.Hg it was 24 L./min./m². This is 14 L./min./m² over the obtained ventilation of 10 L./min./m², except for the ventilation increase caused by exercise itself. This difference was already mentioned as “excess of ventilatory stimuli” and used as an index of the inappropriateness between ventilatory derives of the respiratory center and ventilation achieved by the lung-thorax system. Moreover, if the inappropriateness theory might be considered to admit the above-mentioned gas exchange impairment and the demand for oxygen with this ventilatory function, it
would be more perfect that the grade of consciousness of dyspnea was objectively valued.

Conclusion:

The increase of ventilatory drives is caused not only by disturbances of blood gases but also by reflexes based on the reduction of compliance, increase in pleural pressure and the disorder of lung circulation, and may be influenced also by psychic factors. On the contrast, it is known that these drives decrease in admission of oxygen, sedativa, morphine and so on. It seems that they influence the unbalanced relation, the consciousness of dyspnea, between ventilatory stimuli and the respiratory function.

In the last, when the disturbance of blood gases do cause dyspnea, their threshold values may affect the brain to be mental block and the heart to coronary insufficiency. Therefore, the situation gives very important alarm to the life, should it be emphasized.